

09900336

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FULL ESTIMATED COST	8.70	9.12

=> d l1 1-3

L1 ANSWER 1 OF 3 REGISTRY COPYRIGHT 2003 ACS
RN 258492-26-1 REGISTRY
CN **Oxidoreductase (Streptomyces albus strain ATCC21838 gene sitS)**
(9CI) (CA INDEX NAME)

OTHER NAMES:

CN GenBank AF145724-derived protein GI 5669916
FS PROTEIN SEQUENCE
DR 398008-29-2
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
3 REFERENCES IN FILE CA (1962 TO DATE)
3 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L1 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2003 ACS
RN 230611-74-2 REGISTRY
CN **DNA (Streptomyces albus strain ATCC21838 integrase gene plus open reading frame orf2 plus gene sitI plus gene sitS plus gene sitR plus flanks)** (9CI) (CA INDEX NAME)

OTHER NAMES:

CN GenBank AF145724
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: BIOSIS, CA, CAPLUS, GENBANK

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
2 REFERENCES IN FILE CA (1962 TO DATE)
2 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L1 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2003 ACS
RN 51023-76-8 REGISTRY
CN **Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI)** (CA INDEX NAME)

3/25/2003

09900336

OTHER NAMES:

CN Disodium 4-acetamido-4'-isothiocyanatostilbene-2,2'-disulfonate

CN **SITS**

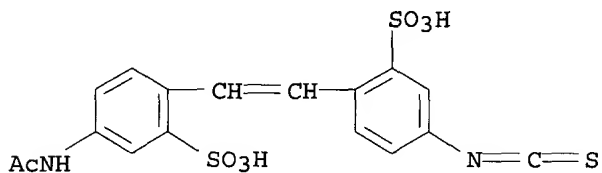
MF C17 H14 N2 O7 S3 . 2 Na

LC STN Files: ADISINSIGHT, AGRICOLA, BIOBUSINESS, BIOTECHNO, CA, CAPLUS,
CHEMCATS, CHEMLIST, CSChem, DDFU, DRUGU, EMBASE, MSDS-OHS, TOXCENTER,
USPATFULL

Other Sources: EINECS**, NDSL**, TSCA**

(**Enter CHEMLIST File for up-to-date regulatory information)

CRN (27816-59-7)



● 2 Na

153 REFERENCES IN FILE CA (1962 TO DATE)

2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

153 REFERENCES IN FILE CAPLUS (1962 TO DATE)

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	8.70	9.12

=> d 11 1-3

L1 ANSWER 1 OF 3 REGISTRY COPYRIGHT 2003 ACS
RN 258492-26-1 REGISTRY
CN Oxidoreductase (Streptomyces albus strain ATCC21838 gene sits)
(9CI) (CA INDEX NAME)

OTHER NAMES:

CN GenBank AF145724-derived protein GI 5669916
FS PROTEIN SEQUENCE
DR 398008-29-2
MF Unspecified
CI MAN
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
3 REFERENCES IN FILE CA (1962 TO DATE)
3 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L1 ANSWER 2 OF 3 REGISTRY COPYRIGHT 2003 ACS
RN 230611-74-2 REGISTRY
CN DNA (Streptomyces albus strain ATCC21838 integrase gene plus open
reading frame orf2 plus gene sitI plus gene sits plus gene sitR plus
flanks) (9CI) (CA INDEX NAME)

OTHER NAMES:

CN GenBank AF145724
FS NUCLEIC ACID SEQUENCE
MF Unspecified
CI MAN
SR GenBank
LC STN Files: BIOSIS, CA, CAPLUS, GENBANK

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
*** USE 'SQD' OR 'SQIDE' FORMATS TO DISPLAY SEQUENCE ***
2 REFERENCES IN FILE CA (1962 TO DATE)
2 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L1 ANSWER 3 OF 3 REGISTRY COPYRIGHT 2003 ACS
RN 51023-76-8 REGISTRY
CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-
sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)

3/25/2003

09900336

OTHER NAMES:

CN Disodium 4-acetamido-4'-isothiocyanatostilbene-2,2'-disulfonate

CN **SITS**

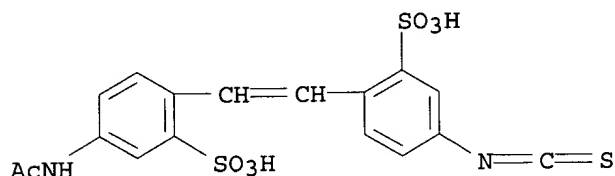
MF C17 H14 N2 O7 S3 . 2 Na

LC STN Files: ADISINSIGHT, AGRICOLA, BIOBUSINESS, BIOTECHNO, CA, CAPLUS,
CHEMCATS, CHEMLIST, CSChem, DDFU, DRUGU, EMBASE, MSDS-OHS, TOXCENTER,
USPATFULL

Other Sources: EINECS**, NDSL**, TSCA**

(**Enter CHEMLIST File for up-to-date regulatory information)

CRN (27816-59-7)



● 2 Na

153 REFERENCES IN FILE CA (1962 TO DATE)

2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

153 REFERENCES IN FILE CAPLUS (1962 TO DATE)

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COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
14.94	15.36

FULL ESTIMATED COST

FILE 'CAPLUS' ENTERED AT 11:44:55 ON 25 MAR 2003

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FILE COVERS 1907 - 25 Mar 2003 VOL 138 ISS 13

FILE LAST UPDATED: 24 Mar 2003 (20030324/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

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MISSING OPERATOR S L1

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

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=> s l1

L2 156 L1

=>

=> d l2 1-5 ibib hitstr abs

L2 ANSWER 1 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2003:133578 CAPLUS

DOCUMENT NUMBER: 138:131069

TITLE: Drug screening for treatment of heart diseases

INVENTOR(S): Okada, Yasunobu; Tanabe, Shigeru

PATENT ASSIGNEE(S): Chugai Seiyaku Kabushiki Kaisha, Japan

SOURCE: PCT Int. Appl., 39 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003014727	A1	20030220	WO 2002-JP8069	20020807
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

PRIORITY APPLN. INFO.:

JP 2001-240852	A	20010808
JP 2001-353047	A	20011119
JP 2002-92363	A	20020328

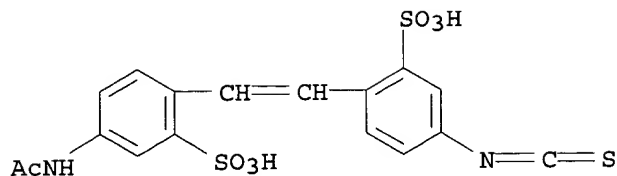
IT 51023-76-8, SITS

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(drug screening by inhibiting apoptosis in heart and vascular cell cultures for treatment of heart diseases)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfonylphenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB It is intended to provide a method of searching for a substance capable of selectively inhibiting apoptosis in heart/vascular cells. The above object can be achieved by providing a method of screening a remedy for heart diseases and/or a preventive for heart diseases characterized by comprising: the step of inducing apoptosis of cultured heart muscular cells and/or cultured vascular endothelial cells; the step of treating the cells with a Cl⁻ channel inhibitor which is a test substance; and the step of evaluating the therapeutic and/or preventive effects of the test substance on heart diseases based on its effect of inhibiting the apoptotic cell death of the muscular cells and/or vascular endothelial cells.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:663788 CAPLUS

DOCUMENT NUMBER: 138:120394

TITLE: Endogenous KV channels in human embryonic kidney (HEK-293) cells

AUTHOR(S): Jiang, Bo; Sun, Xianfeng; Cao, Kun; Wang, Rui

CORPORATE SOURCE: Department of Physiology, University of Saskatchewan, Saskatoon, SK, Can.

SOURCE: Molecular and Cellular Biochemistry (2002), 238(1&2), 69-79

CODEN: MCBIB8; ISSN: 0300-8177

PUBLISHER: Kluwer Academic Publishers

DOCUMENT TYPE: Journal

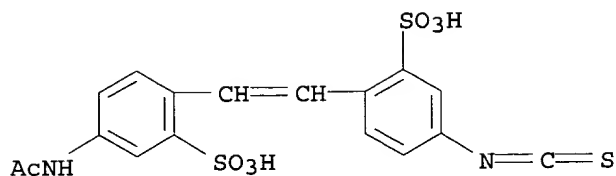
LANGUAGE: English

IT 51023-76-8, SITS

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(multiple endogenous KV genes are expressed in native HEK-293 cells, which possessed endogenous IK and IA currents with unique pharmacol. properties)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB The human embryonic kidney cells (HEK-293) have been widely used as one mammalian expression system in the study of voltage-gated K⁺ (KV) channels. Understanding the endogenous KV channels in these cells is the prerequisite for the characterization of the heterogeneously expressed KV channels in these cells. In the present study we screened the transcriptional expression of different KV genes in HEK-293 cells using reverse transcribed DNA polymerase chain reaction (RT-PCR) method. Among 16 KV genes examd. in native HEK-293 cells 10 KV genes were reproducibly amplified, including those Kv a subunits encoding for the delayed rectifier (IK) [KV1.1, KV1.2, KV1.3, KV1.6, and KV3.1], and for the transient outward KV channels (IA) [KV1.4, KV3.3, KV3.4, and KV4.1] as well as a KVb2. subunit. The whole-cell outward rectifier IK currents in the native HEK-293 cells were recorded (203.±.13 pA at +30 mV, n = 82) with the patch-clamp technique. In about 42% of the examd. cells, IA coexisted with IK currents. IK currents were inhibited by tetraethylammonium chloride (TEA) at 1 and 10 mM by 39.5 and 48.4%, resp. A 39.6% inhibition of IK currents was also obsd. in the presence of 4-aminopyridine (4-AP, 5 mM). Interestingly, both TEA and 4-AP also inhibited IA currents. 4-Acelamido-4'-isothiocyantostilbene-2, 2'-disulfonic acid (1 mM), a Cl channel blocker, had no effect on the endogenous outward currents. We concluded that multiple endogenous KV genes were expressed in native HEK-293 cells, which possessed significant endogenous IK and IA currents with unique pharmacol. properties.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:576636 CAPLUS

DOCUMENT NUMBER: 137:274281

TITLE: The Vibrio cholerae hemolysin anion channel is required for cell vacuolation and death

AUTHOR(S): Moschioni, Monica; Tombola, Francesco; De Bernard, Marina; Coelho, Ana; Zitzer, Alexander; Zoratti, Mario; Montecucco, Cesare

CORPORATE SOURCE: Centro CNR Biomembrane and Dipartimento di Scienze Biomediche Sperimentali, Universita di Padova, Padua, 35121, Italy

SOURCE: Cellular Microbiology (2002), 4(7), 397-409

CODEN: CEMIF5; ISSN: 1462-5814

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

IT 51023-76-8, SITS

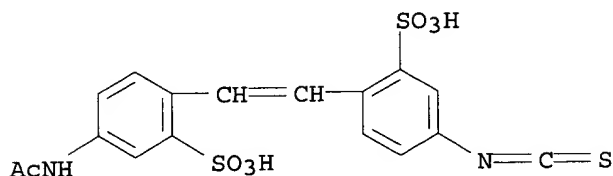
RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors of Vibrio cholerae hemolysin anion channel)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanto-2-

09900336

sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB Several strains of *Vibrio cholerae* secrete a hemolytic toxin of 63 kDa, termed *V. cholerae* cytolysin (VCC). This toxin causes extensive vacuolation and death of cells in culture and forms an anion-selective channel in planar lipid bilayers and in cells. Here, we identify inhibitors of the VCC anion channel and show that the formation of the anion channel is necessary for the development of the vacuoles and for the cell death induced by this toxin. Using markers of cell organelles, we show that vacuoles derive from different intracellular compartments and we identify the contribution of late endosomes and of the trans-Golgi network in vacuole biogenesis.

REFERENCE COUNT: 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:409273 CAPLUS

DOCUMENT NUMBER: 137:722

TITLE: Use of CLC3 chloride channel blockers to modulate vascular tone

INVENTOR(S): Lamb, Fred S.; Schutte, Brian C.; Yang, Baoli

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U. S. Ser. No. 512,926.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002065325	A1	20020530	US 2001-930105	20010815
WO 2003015614	A2	20030227	WO 2002-US26120	20020815
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

PRIORITY APPLN. INFO.: US 1999-121727P P 19990226

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US 2000-512926 A2 20000225
US 2001-930105 A 20010815

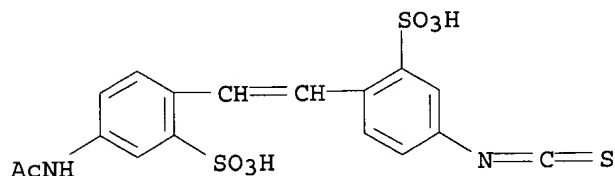
OTHER SOURCE(S): MARPAT 137:722

IT 51023-76-8, SITS

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(use of CLC3 chloride channel blockers to modulate vascular tone)

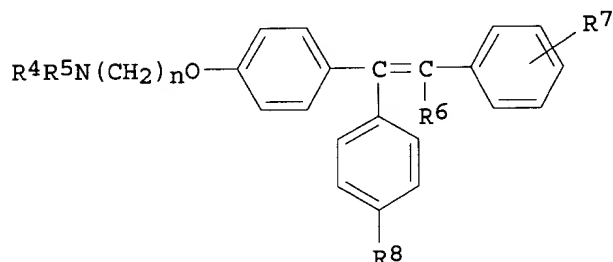
RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

GI



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AB The invention discloses the use of chloride channel blocking compd. I (R4= H, lower alkyl radical; R5= lower alkyl radical; or R4 and R5 connected with adjacent nitrogen to form a heterocyclic radical; R6= H, lower alkyl radical; R7=H, halogen, OH, lower alkyl radical, buta-1-3-dienyl radical which together with adjacent Ph forms a naphthyl radical; R8=H, OH; n=2) for the modulation of vascular tone in a patient having compromised vascular tissue. The present invention also provides methods for the modulation of vascular tone in a patient having compromised vascular tissue, with the administration of a chloride channel blocking agent or a pharmaceutically acceptable salt thereof.

L2 ANSWER 5 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:218864 CAPLUS

DOCUMENT NUMBER: 137:103847

TITLE: Inhibition of Gap junction hemichannels by chloride channel blockers

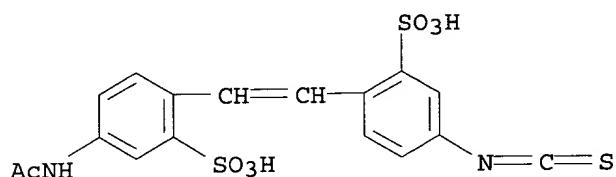
AUTHOR(S): Eskandari, S.; Zampighi, G. A.; Leung, D. W.; Wright, E. M.; Loo, D. D. F.

CORPORATE SOURCE: Department of Physiology, School of Medicine,
Department of Neurobiology, University of California,

3/25/2003

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LosAngeles, CA, 90095-1751, USA
SOURCE: Journal of Membrane Biology (2002), 185(2), 93-102
CODEN: JMBBBO; ISSN: 0022-2631
PUBLISHER: Springer-Verlag New York Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
IT 51023-76-8, SITS
RL: PAC (Pharmacological activity); BIOL (Biological study)
(inhibition of Gap junction hemichannels by chloride channel blockers)
RN 51023-76-8 CAPLUS
CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB Electrophysiol. methods were used to assess the effect of chloride-channel blockers on the macroscopic and microscopic currents of mouse connexin50 (Cx50) and rat connexin46 (Cx46) hemichannels expressed in *Xenopus laevis* oocytes. Oocytes were voltage-clamped at -50 mV and hemichannel currents (ICx50 or ICx46) were activated by lowering the extracellular Ca2+ concn. ([Ca2+]o) from 5 mM to 10 .mu.M. Ion-replacement expts. suggested that ICx50 is carried primarily (>95%) by monovalent cations (PK: PNa: PCl = 1.0: 0.74: 0.05). ICx50 was inhibited by 18.beta.-glycyrrhetic acid (apparent Ki, 2 .mu.M), gadolinium (3 .mu.M), flufenamic acid (3 .mu.M), niflumic acid (11 .mu.M), NPPB (15 .mu.M), diphenyl-2-carboxylate (26 .mu.M), and octanol (177 .mu.M). With the exception of octanol, niflumic acid, and diphenyl-2-carboxylate, the above agents also inhibited ICx46. Anthracene-9-carboxylate, furosemide, DIDS, SITS, IAA-94, and tamoxifen had no inhibitory effect on either ICx50 or ICx46. The kinetics of ICx50 inhibition were not altered at widely different [Ca2+]o (10-500 .mu.M), suggesting that drug-hemichannel interaction does not involve the Ca2+ binding site. In excised membrane patches, application of flufenamic acid or octanol to the extracellular surface of Cx50 hemichannels reduced single channel-open probability without altering the single-channel conductance, but application to the cytoplasmic surface had no effect on the channels. We conclude that some chloride-channel blockers inhibit lens-connexin hemichannels by acting on a site accessible only from the extracellular space, and that drug-hemichannel interaction involves a high-affinity site other than the Ca2+ binding site.

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L2 ANSWER 20 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:602841 CAPLUS

DOCUMENT NUMBER: 131:297859

TITLE: Effects of anion channel antagonists in canine colonic myocytes: comparative pharmacology of Cl⁻, Ca²⁺ and K⁺ currents

AUTHOR(S): Dick, Gregory M.; Kong, In Deok; Sanders, Kenton M.

CORPORATE SOURCE: Department of Physiology & Cell Biology, University of Nevada School of Medicine, Reno, NV, 89557, USA

SOURCE: British Journal of Pharmacology (1999), 127(8), 1819-1831

CODEN: BJPCBM; ISSN: 0007-1188

PUBLISHER: Stockton Press

DOCUMENT TYPE: Journal

LANGUAGE: English

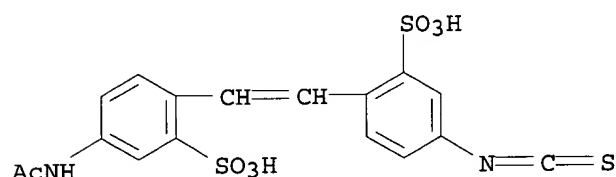
IT 51023-76-8, SITS

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(comparative pharmacol. of chloride, calcium, and potassium currents in colon muscle: channel antagonists as tools)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

AB 1 Vol.-Sensitive, Outwardly Rectifying (VSOR) Cl⁻ currents were measured in canine colonic myocytes by whole-cell patch clamp. Decreasing extracellular osmolarity 50 milliosmoles l-1 activated current that was carried by Cl⁻ and 5-7 times greater in the outward direction. 2 Niflumic acid, an inhibitor of Ca²⁺-activated Cl⁻ channels, did not inhibit VSOR Cl⁻ current. Glibenclamide, an antagonist of CFTR, and anthracene-9-carboxylate (9-AC) inhibited current less than 25% at 100 .mu.M. 3 DIDS (4,4-diisothiocyanato-stilbene-2,2'-disulfonate) inhibited VSOR Cl⁻ current more potently than SITS (4-acetamido-4'-isothiocyanato-stilbene-2,2'-disulfonate). IC₅₀s were 0.84 and 226 .mu.M, resp. 4 VSOR Cl⁻ current was strongly inhibited by tamoxifen ([Z]-1-[p-dimethylaminoethoxy-phenyl]-1,2-diphenyl-1-butene), an anti-estrogen compd. (IC₅₀=0.57 .mu.M). 5 Gd³⁺ antagonized VSOR Cl⁻ current more potently than La³⁺. The IC₅₀ for Gd³⁺ was 23 .mu.M. In contrast, 100 .mu.M La³⁺ inhibited current only 35.+-.7%. 6 Antagonists of VSOR Cl⁻ current had non-specific effects. These compds. blocked voltage-dependent K⁺ and Ca²⁺ currents in colonic myocytes. Tamoxifen (10 .mu.M) and DIDS (10 .mu.M) inhibited L-type Ca²⁺ current 87.+-.7 and 31.+-.5%, resp.

Addnl., in the presence of 300 nM charybdotoxin, tamoxifen (1 .mu.M) and DIDS (10 .mu.M) inhibited delayed rectifier K⁺ current 38.+-.8 and 10.+-.2%, resp. 7 The pharmacol. of VSOR Cl⁻ channels overlaps with voltage-dependent cation channels. DIDS and tamoxifen inhibited VSOR Cl⁻ equally. However, because DIDS had much less effect on L-type Ca²⁺ and delayed rectifier K⁺ channels than did tamoxifen, it might be useful in expts. to investigate the physiol. and pathophysiol. role of this conductance in whole tissues.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 21 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:620052 CAPLUS

DOCUMENT NUMBER: 129:328838

TITLE: Involvement of Stretch-Activated Cl⁻ Channels in Ramification of Murine Microglia

AUTHOR(S): Eder, Claudia; Klee, Rolf; Heinemann, Uwe

CORPORATE SOURCE: Department of Neurophysiology, Institute of Physiology, Humboldt University, Berlin, D 10117, Germany

SOURCE: Journal of Neuroscience (1998), 18(18), 7127-7137
CODEN: JNRSDS; ISSN: 0270-6474

PUBLISHER: Society for Neuroscience

DOCUMENT TYPE: Journal

LANGUAGE: English

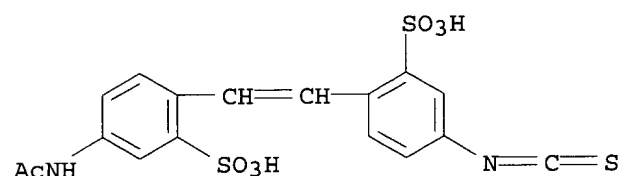
IT 51023-76-8, SITS

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)

(stretch-activated chloride channels in ramification of murine microglia)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetilamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

AB A stretch-activated Cl⁻ current (ICl) was investigated in cultured murine microglia using the whole-cell configuration of the patch-clamp technique. After application of membrane stretch, a Cl⁻ current appeared with seconds, and its amplitude increased further within 3-8 min. The ICl underwent rundown, which was prevented by addn. of 4 mM ATP to the intracellular perfusing soln. The stretch-activated Cl⁻ current exhibited outward rectification and did not show any voltage-dependent gating. Lowering the concn. of extracellular Cl⁻ from 142 to 12 mM by equimolar substitution of Cl⁻ with gluconate shifted the reversal potential of ICl by 41.6 mV in the depolarizing direction. DIDS and SITS blocked ICl in a voltage- and time-dependent manner. At a test potential of +40 mV, a

half-maximal blockade at 16.1 μM DIDS and at 71.0 μM SITS was detd. for ICl. At a concn. of 200 μM , 5-nitro-2-(3-phenylpropylamino)benzoic acid or flufenamic acid blocked ICl by 88% and 75%, resp. Each of these four Cl⁻ channel blockers reversibly inhibited the ramification process of microglia, whereas blockers of voltage-gated Na⁺ and K⁺ channels did not affect the transformation of microglia from their ameboid into the ramified phenotype. It is suggested that in microglia functional stretch-activated Cl⁻ channels are required for the induction of ramification but not for maintaining the ramified shape.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 22 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:548103 CAPLUS

DOCUMENT NUMBER: 129:211495

TITLE: Effects of renal cytoprotective agents on erythrocyte membrane stability

AUTHOR(S): Peters, Susan M. A.; De Jong, Maarten D.; Bindels, Rene J. M.; Van Os, Carel H.; Wetzels, Jack F. M.

CORPORATE SOURCE: Department of Cell Physiology, University of Nijmegen, Nijmegen, 6500 HB, Neth.

SOURCE: Life Sciences (1998), 63(11), 975-983

CODEN: LIFSAK; ISSN: 0024-3205

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

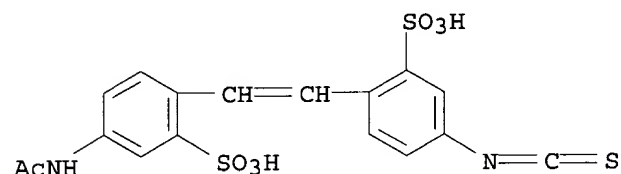
IT 51023-76-8, SITS

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(effects of renal cytoprotective agents on erythrocyte membrane stability)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB To elucidate potential mechanisms of ischemic renal injury, investigators often use drugs that interfere with specific pathol. pathways and study their protective efficacy in in vitro models of ischemia, such as isolated renal proximal tubules subjected to hypoxia. However, the protective effects of certain drugs may depend on non-specific membrane-stabilizing properties. We have studied the effects of several drugs on membrane integrity using osmotic lysis of erythrocytes as a model system. Freshly isolated rabbit erythrocytes were subjected to a hypotonic shock, and the protective effects of various Ca channel blockers, phospholipase inhibitors, free fatty acids, the NO-synthase inhibitor L-NAME, the amino

acid glycine and its receptor-analog strychnine, and 2 Cl channel blockers were examd. Most agents protected erythrocytes against hypotonic hemolysis when added to the medium in the same concn. range as used in suspensions of hypoxic proximal tubules. Only the protective agents that proposedly act via a blockade of Cl influx (glycine, strychnine, and the Cl channel blockers), did not attenuate hypotonic hemolysis. The erythrocyte hemolysis assay may provide an easy and rapid method to screen for non-specific membrane-stabilizing effects of potentially cytoprotective agents.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 23 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:468544 CAPLUS

DOCUMENT NUMBER: 129:185717

TITLE: Component analysis of the fast photoelectric signal from model bacteriorhodopsin membranes V. Effects of chloride ion transport blockers and divalent cation chelators

AUTHOR(S): Petrak, Michelle R.; Hong, Felix T.

CORPORATE SOURCE: Department of Physiology, Wayne State University, School of Medicine, Detroit, MI, 48201, USA

SOURCE: Bioelectrochemistry and Bioenergetics (1998), 45(2), 193-201

CODEN: BEBEBP; ISSN: 0302-4598

PUBLISHER: Elsevier Science S.A.

DOCUMENT TYPE: Journal

LANGUAGE: English

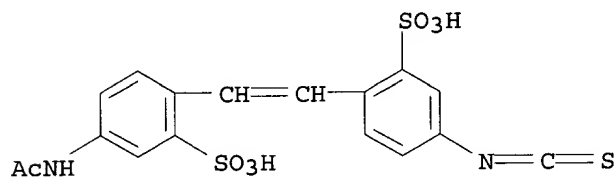
IT 51023-76-8, SITS

RL: BPR (Biological process); BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); BIOL (Biological study); PROC (Process)

(component anal. of fast photoelec. signal from model bacteriorhodopsin membranes, effects of chloride ion transport blockers and divalent cation chelators)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



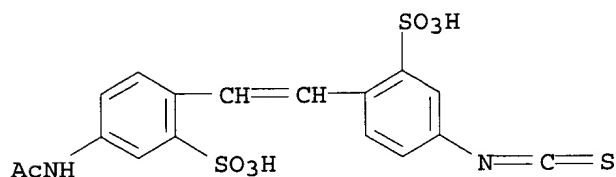
●2 Na

AB Bacteriorhodopsin (BR), the light-driven proton pump found in Halobacteria salinarium, exhibits a fast photoelec. signal which is the manifestation of light-induced vectorial charge sepn. and recombination in the purple membrane. The photosignal can be decompd. into three components (B1, B2, and B2'). We have assocd. these components with chem. processes taking place at various domains of bacteriorhodopsin (B1 from hydrophobic regions, and B2 and B2' from the intracellular and extracellular

hydrophilic domains, resp.). In this report, we investigate the effect of halide ions and divalent cations on the B1 and the B2 components. We found that halide ions are required for the generation of the B2 component at low pH whereas divalent cations enhance the B2 component at medium to high pH. In addn., these signals can be either abolished or inhibited by blockers of chloride ion transport and by divalent cation chelators, resp. We tentatively decomp. the B2 component into two subcomponents: B2-a for the Cl--dependent subcomponent that appears at low pH, and B2-c for the divalent cation-sensitive subcomponent that appears at medium to high pH. It is possible that the B2-a component may be generated by interfacial Cl--transfer whereas the B2-c component may be generated by interfacial proton transfer.

REFERENCE COUNT: 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 24 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1997:438973 CAPLUS
 DOCUMENT NUMBER: 127:106844
 TITLE: The role of eicosanoids and progesterone in ovulation of *Rana temporaria* oocytes
 AUTHOR(S): Skoblina, M. N.; Kondrat'eva, O. T.; Nikiforova, G. P.; Huhtaniemi, I.
 CORPORATE SOURCE: Kol'tsov Institute of Developmental Biology, Russian Academy of Sciences, Moscow, 117808, Russia
 SOURCE: Russian Journal of Developmental Biology (Translation of Ontogenez) (1997), 28(3), 170-175
 CODEN: RJDBE2; ISSN: 1062-3604
 PUBLISHER: MAIK Nauka/Interperiodica
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 IT 51023-76-8, SITS
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
 (eicosanoids and progesterone role in ovulation of *Rana temporaria* oocytes)
 RN 51023-76-8 CAPLUS
 CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)

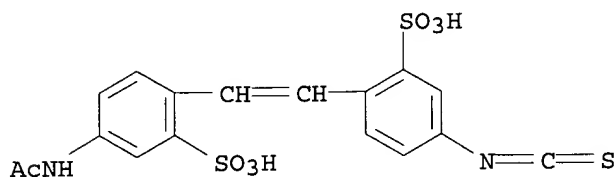


●2 Na

AB Prostaglandin F2.alpha. (1-5 .mu.g/mL) stimulated ovulation in vitro of *Rana temporaria* oocytes in the absence of pituitary suspension and potentiated the effects of progesterone. The inhibitor of cyclooxygenase indomethacin (0.01-10 .mu.g/mL) decreased the rate of oocyte ovulation stimulated by the pituitary suspension. An increased pituitary suspension concn. decreased the inhibitory effect of indomethacin. Indomethacin did not affect oocyte ovulation stimulated by prostaglandin F2.alpha. or

progesterone. The inhibition of ovulation by the chloride channel blocker SITS (10 μ M) is partly relieved by prostaglandin F₂ α . or progesterone but completely eliminated by their mixt.

L2 ANSWER 25 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1997:341237 CAPLUS
 DOCUMENT NUMBER: 127:30519
 TITLE: Stretch-activated current in rabbit sino-atrial node cells
 AUTHOR(S): Hagiwara, Nobuhisa; Tamura, Koji; Shoda, Morio; Matsuda, Naoki; Kajimoto, Katsuya; Sakai, Rieko; Kasanuki, Hiroshi; Hosoda, Saichi
 CORPORATE SOURCE: Heart Inst. Japan, Tokyo Women's Med. Coll., Tokyo, 162, Japan
 SOURCE: Tokyo Joshi Ika Daigaku Zasshi (1997), 67(4), 227-231
 CODEN: TJIZAF; ISSN: 0040-9022
 PUBLISHER: Tokyo Joshi Ika Daigaku Gakkai
 DOCUMENT TYPE: Journal
 LANGUAGE: Japanese
 IT 51023-76-8, SITS
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
 (role of stretch-activated channel in sino-atrial node cells)
 RN 51023-76-8 CAPLUS
 CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



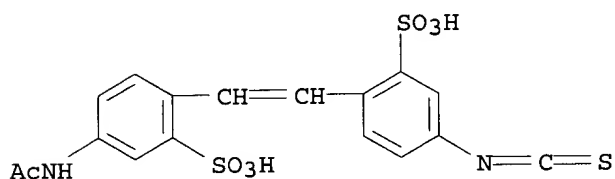
● 2 Na

AB Stretch-activated current was studied in rabbit sino-atrial node cells using the whole-cell patch clamp method. With continuous application of pos. pressure through the patch electrode, the cell was inflated and the membrane conductance was increased. The stretch-activated current showed time-independent and outward rectifying properties and the current was sensitively reduced by the Cl channel blockers, such as SITS, DNDS or 9-AC. The reversal potential of stretch-activated current was well explained by the equil. potential of Cl. These findings indicate that the stretch-activated current is Cl selective, and the results suggested that the stretch-activated Cl current may contribute to the pos. chronotropic effect during mech. stimulation in sino-atrial node cells.

L2 ANSWER 26 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1997:193491 CAPLUS
 DOCUMENT NUMBER: 126:290324
 TITLE: Measurement of the distribution of anion exchange function in normal human red cells
 AUTHOR(S): Raftos, Julia E.; Bookchin, Robert M.; Lew, V. L.
 CORPORATE SOURCE: The Physiological Laboratory, University of Cambridge,

09900336

SOURCE: Cambridge, CB2 3EG, UK
Journal of Physiology (Cambridge, United Kingdom)
(1997), 499(1), 17-25
CODEN: JPHYA7; ISSN: 0022-3751
PUBLISHER: Cambridge University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
IT 51023-76-8, Sits
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
(Uses)
(measurement of distribution of anion exchange function in normal human
red cells)
RN 51023-76-8 CAPLUS
CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-
sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB The aim of the present work was to investigate cell-to-cell variation in anion exchange turnover in normal human red cells. Red cells permeabilized to protons and K⁺ dehydrate extremely rapidly by processes that are rate-limited by the induced K⁺ permeability or by anion exchange turnover. Conditions were designed to render dehydration rate-limited by anion exchange turnover. Cell-to-cell variation in anion exchange function could then be measured from the distribution of delay times required for dehydrating cells to attain resistance to hemolysis in a selected hypotonic medium. Red cells were suspended at 10% hematocrit in a low-K⁺ soln. and, after a brief pre-incubation with 20 .mu.M SITS at 4.degree., were warmed to 24.degree., and the protonophore CCCP was added (20 .mu.M) followed 2 min later by valinomycin (60 .mu.M). Delay times for cells to become resistant to lysis were measured from the instant of valinomycin addn. by sampling suspension aliquots into thirty vols. of 35 mM NaCl. After centrifugation the per cent lysis was estd. by measuring the Hb concn. in the supernatant. Typical median delay times with this standardized method were 4-5 min. The statistical parameters of the delay time distributions report the population spread in the transport function that was limiting to dehydration. In the absence of SITS and CCCP, dehydration was limited by the diffusional Cl⁻ permeability (P_{Cl}). Delay time distributions for P_{Cl}- and anion exchange-limited dehydration were measured in red cells from three normal donors. For both distributions, the coeffs. of variation ranged between 13.0 and 15.2%, indicating a high degree of uniformity in P_{Cl} and anion exchange function among individual red cells.

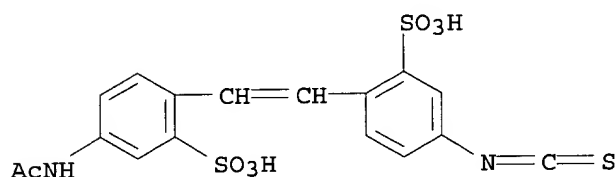
L2 ANSWER 27 OF 156 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:192410 CAPLUS
DOCUMENT NUMBER: 126:262222
TITLE: Effect of anion transport inhibitors on hemolysis

3/25/2003

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induced by melittin
AUTHOR(S): Kurbanazarova, R. Sh.; Krasil'nikov, O. V.; Kragoe, E.
D.; Sabirov, R. Z.
CORPORATE SOURCE: Inst. Fiziol. i Biofiz., AN RUz, Uzbekistan
SOURCE: Doklady Akademii Nauk Respubliki Uzbekistan (1996),
(6), 47-49
CODEN: DARUEE
PUBLISHER: Fan
DOCUMENT TYPE: Journal
LANGUAGE: Russian

IT 51023-76-8, SITS
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or
effector, except adverse); BSU (Biological study, unclassified); BIOL
(Biological study)
(effect of anion transport inhibitors on hemolysis induced by melittin)
RN 51023-76-8 CAPLUS
CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-
sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB The effects of four anion transport inhibitors [DIDS, SITS, B-3(+) and
IIA-94(+)] on melittin induced hemolysis were investigated. At high
concns. DIDS and SITS inhibited but B-3(+) and IIA-94(+) facilitated the
hemolytic effect of melittin. At low concns. of inhibitors their effects
on melittin-induced hemolysis were opposite. The inhibitors were actually
able to change the melittin-induced hemolysis but these effects were not a
result of damage of the anion transport system of erythrocytes.

L2 ANSWER 28 OF 156 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:644845 CAPLUS
DOCUMENT NUMBER: 125:265576
TITLE: Effects of stilbene derivatives SITS and DIDS on
development of intracellular acidosis during ischemia
in isolated guinea pig ventricular papillary muscle in
vitro
AUTHOR(S): Lai, Zhong-Fang; Liu, Jie; Nishi, Katsuhide
CORPORATE SOURCE: Department of Pharmacology, Kumamoto University School
of Medicine, Kumamoto, 860, Japan
SOURCE: Japanese Journal of Pharmacology (1996), 72(2),
161-174
CODEN: JJPAAZ; ISSN: 0021-5198
PUBLISHER: Japanese Pharmacological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
IT 51023-76-8, SITS
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); BIOL (Biological study)

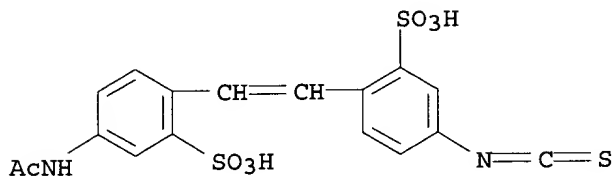
3/25/2003

09900336

(acidosis during ischemia in ventricular papillary muscle response to)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetlamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

AB Ion-selective microelectrode techniques were used to investigate the effects of 4-acetamido-4'-isothiocyanatostilbene-2,2'-disulfonic acid (SITS) and 4,4'-diisothiocyanatostilbene-2,2'-disulfonic acid (DIDS), known as Cl--HCO₃- exchange blockers, on action potentials and intracellular pH (pHi) in guinea pig ventricular papillary muscles subjected to simulated ischemia. Simulated ischemia was produced by stopping the flow of superfusing soln. and then covering the prepns. with mineral oil. Simulated ischemia induced a progressive decrease in the max. upstroke rate and resting membrane potentials, shortened action potential duration, and resulted in cessation of action potentials within 10-12 min after the onset of simulated ischemia. The pHi measurements revealed progressive intracellular acidosis during the period of simulated ischemia. SITS (0.5 mM) or DIDS (0.1 mM) delayed the onset of ischemia-induced deterioration of action potentials and prolonged the time to cessation of action potentials. SITS or DIDS (0.1-0.5 mM) induced an increase in pHi in HCO₃--buffered soln. and suppressed the development of intracellular acidosis during ischemia. Under external Cl--free conditions, the time to cessation of action potentials caused by ischemia was delayed, and the development of intracellular acidosis during ischemia was attenuated. The results indicate that activation of the Cl--HCO₃- exchange system may be involved, in part, in the development of intracellular acidosis during cardiac ischemia.

L2 ANSWER 29 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:565085 CAPLUS

DOCUMENT NUMBER: 125:217191

TITLE: Inorganic carbon uptake for photosynthesis by the symbiotic coral-dinoflagellate association. II. Mechanisms for bicarbonate uptake

AUTHOR(S): Al-Moghrabi, Salim; Goiran, Claire; Allemand, Denis; Speziale, Nathalie; Jaubert, Jean

CORPORATE SOURCE: Observatoire Oceanologique Europeen, Centre Scientifique de Monaco, Monaco, MC-98000, Monaco

SOURCE: Journal of Experimental Marine Biology and Ecology (1996), 199(2), 227-248
CODEN: JEMBAM; ISSN: 0022-0981

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

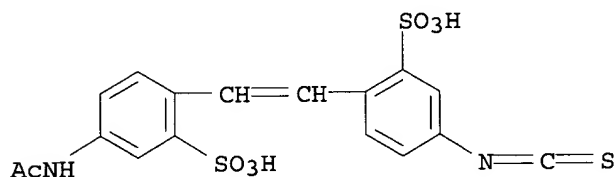
IT 51023-76-8, SITS

RL: BAC (Biological activity or effector, except adverse); BSU (Biological

study, unclassified); BIOL (Biological study)
 (mechanism of dissolved inorg. carbon transport by the symbiotic
 coral-dinoflagellate assocn.)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB Mechanisms of HCO₃⁻ uptake as a source of dissolved inorg. carbon (DIC) for photosynthesis by the intracellular symbiont, *Symbiodinium* sp. were studied using microcolonies of the coral *Galaxea fascicularis*, freshly isolated zooxanthellae (FIZ) and cultured zooxanthellae (CZ). For this purpose specific inhibitors of anion transport 4-acetamido-4'-isothiocyanatostilbene-2,2'-disulfonic acid - SITS -, 4,4'-diisothiocyanato-stilbene-2,2'-disulfonic acid - DIDS -, carbonic anhydrase (acetazolamide, ethoxycarbonylamilofylline), H⁺-ATPase (N,N'-dicyclohexylcarbodiimide - DCCD -, diethylstilbestrol - DES -, vanadate) or Ca²⁺ channels (verapamil) were used. The effect of ions known to play a role in HCO₃⁻ transport, like Na⁺ and Ca²⁺ were also tested. Chloride uptake expts. were also performed to det. whether Cl⁻ and HCO₃⁻ fluxes were coupled in CZ. Furthermore, the presence of carbonic anhydrase was tested using indirect immunofluorescence. The results suggest that bicarbonate uptake by the animal symbiont is likely to be achieved by two types of DIDS-sensitive HCO₃⁻ carriers, each sharing 50% of the total uptake. The first is Na⁺-dependent, while the second is Na⁺-independent. It is suggested that the presence of a Na⁺-independent Cl⁻/HCO₃⁻ exchange and either a Na⁺-dependent Cl⁻/HCO₃⁻ exchange or a Na⁺/HCO₃⁻ symport. Pharmacol. data suggest that the enzyme carbonic anhydrase plays an important role in maintaining the photosynthetic rate. In the intact symbiosis, the major fraction of carbonic anhydrase activity is located in the zooxanthellae. Striking differences in DIC absorption mechanisms were found for FIZ and CZ. In FIZ, H⁺-ATPase and carbonic anhydrase participate in the carbon supply while in CZ the mechanism of HCO₃⁻ uptake appears to be strictly Na⁺-dependent and could be the result of Na⁺/HCO₃⁻ symport activity. It is hypothesized that stimulation of HCO₃⁻ uptake by the animal host is a consequence of intracellular pH alkalization by zooxanthellae photosynthesis. These results were summarized in a synthetic scheme of DIC absorption by both host cell and isolated zooxanthellae.

L2 ANSWER 30 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:496092 CAPLUS

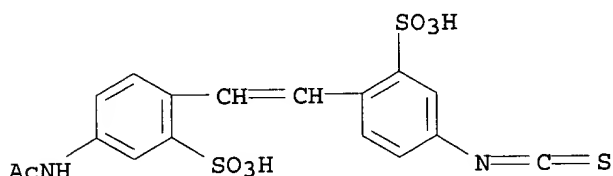
DOCUMENT NUMBER: 125:185368

TITLE: Use of chloride blockers: a novel approach for cardioprotection against ischemia-reperfusion damage

AUTHOR(S): Tanaka, Hikaru; Matsui, Saiko; Kawanishi, Toru; Shigenobu, Koki

09900336

CORPORATE SOURCE: Sch. Pharm. Sci., Tohoku Univ., Funabashi, Japan
SOURCE: Journal of Pharmacology and Experimental Therapeutics
(1996), 278(2), 854-861
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
IT 51023-76-8, SITS
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of chloride blockers as a novel approach for cardioprotection against ischemia-reperfusion damage)
RN 51023-76-8 CAPLUS
CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB We examd. whether the chloride channel blockers anthracene-9-carboxylic acid (9-AC) and 4-acetamide-4'-isothiocyanatostilbene-2,2'-disulfonic acid (SITS) exert protective effects against myocardial ischemia-reperfusion damage. In isolated guinea pig ventricular cells, 9-AC (200 .mu.M), but not SITS (100 .mu.M), inhibited the chloride current induced by isoproterenol. Elec. and mech. activities and intracellular pH of arterially perfused guinea pig right ventricular preps. were recorded with an intracellular microelectrode, a force transducer and a pH-sensitive fluorescent probe, resp. The preps. were subjected to 30 min of no-flow ischemia, with or without 9-AC (100 .mu.M) or SITS (1 .mu.M), followed by reperfusion. No flow ischemia produced decreases in action potential amplitude and duration, and contractile force was completely abolished. Although the changes in elec. parameters were reversed upon reperfusion, the contractile force recovered only to about 50% of preischemic values. 9-AC and SITS had no inhibitory effect on contractile force under normal conditions and during ischemia but significantly improved the recovery of contractile force upon reperfusion to about 80% of preischemic values. Both 9-AC and SITS showed significant inhibition of the ischemia-induced abbreviation of action potential duration. Other parameters were not affected by 9-AC or SITS. During ischemia, intracellular pH showed a transient small increase followed by a sustained decrease, which was completely recovered upon reperfusion. The decrease in pH during ischemia was attenuated by 80% in SITS-but not 9-AC-treated preps. Thus, we demonstrated that the chloride channel blockers 9-AC and SITS, which have no cardiosuppressive effects, exert protective effects against myocardial ischemia-reperfusion damage.

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=> d 12 150-155

L2 ANSWER 150 OF 156 CAPLUS COPYRIGHT 2003 ACS
AN 1979:588072 CAPLUS
DN 91:188072
TI A voltage-gated anion channel from the electric organ of Torpedo californica
AU White, Michael M.; Miller, Christopher
CS Grad. Dep. Biochem., Brandeis Univ., Waltham, MA, 02154, USA
SO Journal of Biological Chemistry (1979), 254(20), 10161-6
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English

L2 ANSWER 151 OF 156 CAPLUS COPYRIGHT 2003 ACS
AN 1979:432693 CAPLUS
DN 91:32693
TI A new class of drugs that inhibit platelet release and aggregation
AU Shulman, N. Raphael; Pollard, Harvey B.; Tack-Goldman, Karen; Buda, Edwarda
CS Clin. Hematol. Branch, NIH, Bethesda, MD, USA
SO Transactions of the Association of American Physicians (1978), 91, 104-17
CODEN: TAAPAI; ISSN: 0066-9458
DT Journal
LA English

L2 ANSWER 152 OF 156 CAPLUS COPYRIGHT 2003 ACS
AN 1979:162649 CAPLUS
DN 90:162649
TI Effects of a disulfonic stilbene SITS on anion reabsorption from the proximal tubule of the rat
AU Bishop, J. H. V.; Green, R.
CS Dep. Physiol., Univ. Manchester, Manchester, UK
SO Journal of Physiology (Cambridge, United Kingdom) (1978), 285, 42P
CODEN: JPHYA7; ISSN: 0022-3751
DT Journal
LA English

L2 ANSWER 153 OF 156 CAPLUS COPYRIGHT 2003 ACS
AN 1978:502092 CAPLUS
DN 89:102092
TI Permeation of the erythrocyte stroma by superoxide radical
AU Lynch, Robert E.; Fridovich, Irwin
CS Dep. Med., Univ. Utah Coll. Med., Salt Lake City, UT, USA
SO Journal of Biological Chemistry (1978), 253(13), 4697-9
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English

L2 ANSWER 154 OF 156 CAPLUS COPYRIGHT 2003 ACS
AN 1978:502052 CAPLUS
DN 89:102052
TI Evidence for stimulation of anion transport in ATP-evoked transmitter release from isolated secretory vesicles
AU Pazoles, Christopher J.; Pollard, Harvey B.
CS Natl. Inst. Child Health Human Dev., Bethesda, MD, USA
SO Journal of Biological Chemistry (1978), 253(11), 3962-9
CODEN: JBCHA3; ISSN: 0021-9258

3/25/2003

09900336

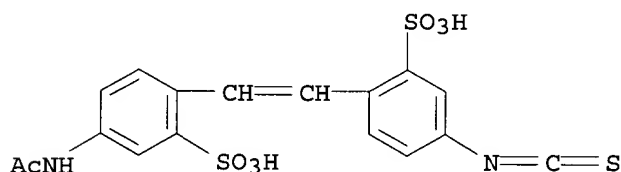
DT Journal
LA English

L2 ANSWER 155 OF 156 CAPLUS COPYRIGHT 2003 ACS
AN 1978:500892 CAPLUS
DN 89:100892
TI Inhibition of the bicarbonate exit step in urinary acidification by a
disulfonic stilbene
AU Cohen, Loren H.; Mueller, Allan; Steinmetz, Philip R.
CS Dep. Med., Univ. Iowa Coll. Med., Iowa City, IA, USA
SO Journal of Clinical Investigation (1978), 61(4), 981-6
CODEN: JCINAO; ISSN: 0021-9738
DT Journal
LA English

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=> d l2 140-145 ibib hitstr abs

L2 ANSWER 140 OF 156 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1981:547782 CAPLUS
DOCUMENT NUMBER: 95:147782
TITLE: Sulfate transport in rabbit proximal convoluted
tubules: presence of anion exchange
AUTHOR(S): Brazy, Peter C.; Dennis, Vincent W.
CORPORATE SOURCE: VA Med. Cent., Duke Univ., Durham, NC, 27710, USA
SOURCE: American Journal of Physiology (1981), 241(3),
F300-F307
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English
IT 51023-76-8
RL: BIOL (Biological study)
(sulfate transport by kidney in response to)
RN 51023-76-8 CAPLUS
CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-
sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



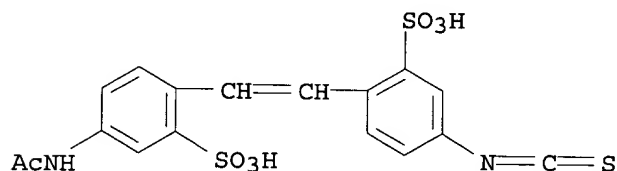
●2 Na

AB The characteristics of SO₄²⁻ transport in proximal convoluted tubules from rabbit kidney are described. Absorptive and secretory fluxes of SO₄²⁻ were measured in isolated tubular segments perfused and bathed with fluids contg. SO₄²⁻ concns. of 0.2-10 mM. At 2.0 mM, the SO₄²⁻ flux in the absorptive direction averaged 4.76 and the secretory flux was 3.08 pnmol/mm/min. Ouabain 10⁻⁵M decreased each to .apprx.1.15 pmol/mm/min. Kinetic anal. of each unidirectional SO₄²⁻ flux demonstrated satn. with increasing SO₄²⁻ concns. S₂O₃²⁻ (2 mM) in the bath inhibited both

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absorptive and secretory SO₄²⁻ fluxes; S₂O₃²⁻ in the perfusate inhibited only the absorptive flux. Similar results were obtained with 10⁻⁶M SITS in either bath or perfusate. Phosphate had no effect on SO₄²⁻ transport. Each unidirectional SO₄²⁻ flux was influenced by the SO₄²⁻ concn. in the soln. on the opposite side in a pattern consistent with the presence of an anion exchange mechanism. Anion-exchange transport persisted at 22.degree. when net SO₄²⁻ transport was abolished. Evidently, SO₄²⁻ transport in the proximal convoluted tubule is bidirectional, independent of phosphate transport, and occurs via 2 forms of facilitated transport, 1 of which is an anion-exchange mechanism.

L2 ANSWER 141 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1981:168584 CAPLUS
 DOCUMENT NUMBER: 94:168584
 TITLE: Effects of amiloride and SITS on branchial ion fluxes in rainbow trout, *Salmo gairdneri*
 AUTHOR(S): Perry, S. F.; Randall, D. J.
 CORPORATE SOURCE: Dep. Zool., Univ. British Columbia, Vancouver, BC, V6T 2A9, Can.
 SOURCE: Journal of Experimental Zoology (1981), 215(2), 225-8
 CODEN: JEZOAO; ISSN: 0022-104X
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 IT 51023-76-8
 RL: PRP (Properties)
 (ion transport inhibition by, in rainbow trout)
 RN 51023-76-8 CAPLUS
 CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB Both amiloride [2609-46-3] and SITS [51023-76-8] significantly inhibited branchial influx of Na⁺ and Cl⁻ in rainbow trout. The inhibitory effects on the contralateral exchange processes may result from changes in gill epithelial cell pH.

L2 ANSWER 142 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1980:600492 CAPLUS
 DOCUMENT NUMBER: 93:200492
 TITLE: Cytofluorometric and cytophotometric DNA measurements of cervical smears stained using a new bi-color method
 AUTHOR(S): Ploem-Zaaijer, J. J.; Beyer-Boon, M. E.; Leyte-Veldstra, L.; Ploem, J. S.
 CORPORATE SOURCE: Univ. Leiden, Leiden, Neth.
 SOURCE: Proc. Int. Conf. Autom. Cancer Cytol. Cell Image Anal., 2nd (1979), Meeting Date 1977, 225-35.
 Editor(s): Pressman, Norman J.; Wied, George L.

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Tutorials Cytol.: Chicago, Ill.

CODEN: 44HGAX

DOCUMENT TYPE:

Conference

LANGUAGE:

English

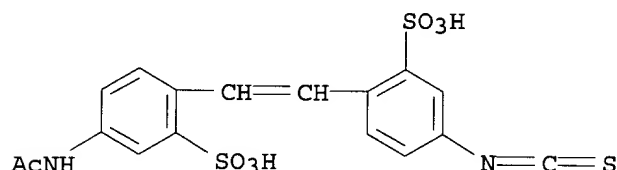
IT 51023-76-8

RL: ANST (Analytical study)

(staining by, of DNA and proteins of cervical smears)

RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



● 2 Na

AB Cervical smears were automatically stained for DNA by an acriflavine-Feulgen procedure and for proteins with SITS. These were compared with Papanicolaou-stained preps. The occurrence of cells with high DNA ploidy values (>5 C) were analyzed using a special microfluorometer that allowed visual screening of the entire prep. as well as quant. fluorescence intensity measurements on single cells. The instrument had 3 light sources for transmitting light at the following 3 wavelengths: 425 nm for obtaining a relatively weak fluorescent image of both nucleus and cytoplasm simultaneously, 480 nm for obtaining an absorbance image of nuclei, and 485 nm for obtaining strong nuclear fluorescence in DNA detns. Nuclei with strongly increased DNA content were present in 96% of 334 cases classified as moderate-severe dysplasia, atypical reserve cell hyperplasia, carcinoma in situ, and invasive carcinoma. Cell morphol.-DNA value relations were discussed.

L2 ANSWER 143 OF 156 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1980:561250 CAPLUS

DOCUMENT NUMBER: 93:161250

TITLE: Effect of an anion transport inhibitor on blood-brain barrier lesions during acute hypertension. Possible prevention of transendothelial vesicular transport
AUTHOR(S): Hardebo, Jan Erik; Johansson, Barbro B.
CORPORATE SOURCE: Dep. Histol. Neurol., Univ. Lund, Lund, S-223 62, Swed.

SOURCE: Acta Neuropathologica (1980), 51(1), 33-8

CODEN: ANPTAL; ISSN: 0001-6322

DOCUMENT TYPE: Journal

LANGUAGE: English

IT 51023-76-8

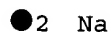
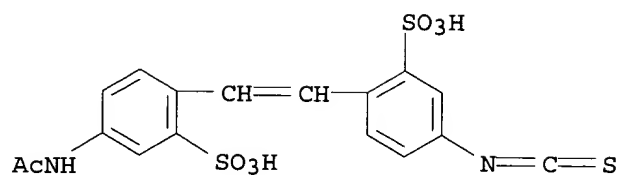
RL: BIOL (Biological study)

(blood-brain barrier lesions during acute hypertension prevention by)

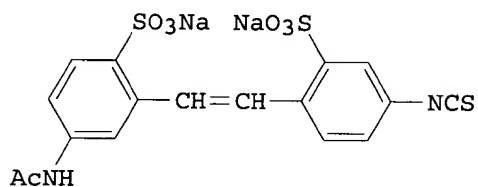
RN 51023-76-8 CAPLUS

CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulphophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)

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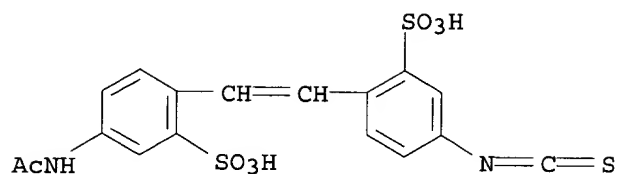
GI



I .

AB SITS (I) [51023-76-8] prevented leakage across the blood-brain barrier (BBB) into the brain parenchyma following a hypertensive insult induced by a local increase of the intraluminal pressure in anesthetized rats and by i.v. administration of adrenaline or bicuculline in conscious unrestrained animals. Since SITS increased cerebral blood flow the protection cannot be explained by a constrictor action on the cerebral vessels. SITS is a drug with complex action on the cell membrane including an inhibitory effect on anion transport mechanisms and on some cyclic AMP-mediated processes. It is possible that the protection of the BBB obsd. in the present study is related to a decrease in cyclic AMP, but a membrane-stabilizing effect can at present not be excluded.

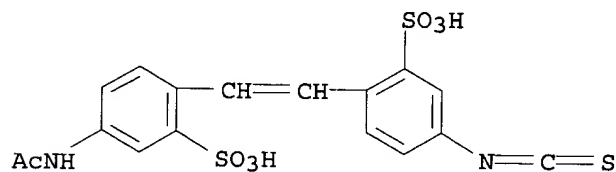
L2 ANSWER 144 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1980:440892 CAPLUS
 DOCUMENT NUMBER: 93:40892
 TITLE: Chloride efflux measurements in mammalian skeletal muscle
 AUTHOR(S): Hayward, B. S.; Barchi, R. L.
 CORPORATE SOURCE: Sch. Med., Univ. Pennsylvania, Philadelphia, PA, 19104, USA
 SOURCE: Muscle & Nerve (1980), 3(3), 207-15
 CODEN: MUNEDE; ISSN: 0148-639X
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 IT 51023-76-8
 RL: ANST (Analytical study)
 (muscle chloride efflux response to)
 RN 51023-76-8 CAPLUS
 CN Benzenesulfonic acid, 5-(acetilamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

AB A rapid sampling technique was used to resolve the components of ^{36}Cl efflux from isolated extensor digitorum longus muscles of young rats. Four distinct fluxes with apparent rate consts. of 4.40 min^{-1} (k_1), 1.30 min^{-1} (k_2), 0.24 min^{-1} (k_3), and 0.048 min^{-1} (k_4) at 30°C were identified. Together, these fluxes accounted for the movement of $>98\%$ of exchangeable muscle Cl^- . The muscle compartment assocd. with the fastest flux (k_1) contained 23% of the total muscle Cl^- corresponding to the extracellular space as detd. with inulin or mannitol. The compartment assocd. with k_2 accounted for 71% of the intracellular vol., and k_2 was assumed to represent ^{36}Cl efflux across the surface membrane. The rate const. k_2 was temp.-dependent with a Q_{10} of 1.11 at $5\text{--}30^\circ\text{C}$. Arom. carboxylic acids known to block sarcolemmal Cl^- conductance specificity lowered k_2 by 25% at 30°C as did replacement of external Cl^- with NO_3^- .

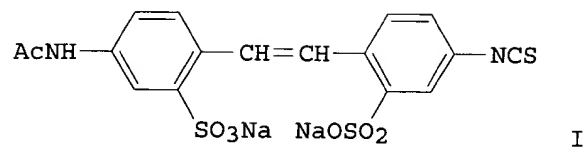
L2 ANSWER 145 OF 156 CAPLUS COPYRIGHT 2003 ACS
 ACCESSION NUMBER: 1980:437090 CAPLUS
 DOCUMENT NUMBER: 93:37090
 TITLE: Effect of SITS on chlorine-36-efflux from the rat proximal convoluted tubule
 AUTHOR(S): Greenwood, S. L.
 CORPORATE SOURCE: Dep. Physiol., Univ. Manchester, Manchester, M13 9PT, UK
 SOURCE: Journal of Physiology (Cambridge, United Kingdom) (1980), 302, 27P-28P
 CODEN: JPHYA7; ISSN: 0022-3751
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 IT 51023-76-8
 RL: BIOL (Biological study)
 (kidney tubules reabsorption of chloride inhibition by, mechanism of)
 RN 51023-76-8 CAPLUS
 CN Benzenesulfonic acid, 5-(acetylamino)-2-[2-(4-isothiocyanato-2-sulfophenyl)ethenyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

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GI



AB Micropuncture studies on kidneys of anesthetized rats showed that SITS (I) [51023-76-8] (1 mM) in the perfusate inhibited fractional reabsorption of ³⁶Cl⁻ by proximal convoluted tubules, measured by comparing the ³⁶Cl⁻ : ³H inulin ratio in recollected perfusate with the initial value. This inhibition occurred when Na was absent from the medium. Thus, the inhibition of I may be due to a direct effect on anion transport.

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